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THE PHYSIOLOGICAL EFFECT OF COMPRESSIVE FORCES ON THE TORSO*

Project X-630

Report No. 8

By

HOWARD R. BIERMAN
Commander, MC, USNR

RUSSELL M. WILDER, JR.
Lieutenant, MC, USNR

and

HARPER K. HELLEMS
Lieutenant (jg), MC, USNR

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INTRODUCTION

Under the stimulus of military aviation the physiological responses of the human body to radial accelerations have been carefully investigated. Until very recently, relatively few investigations have been undertaken to study the physiological problems of linear acceleration. With the advent of jet and rocket propulsion these studies will have added significance.

During the earlier phases of the investigation of deceleration, it became apparent that human subjects would have to be used if experimental results were to be applied with any degree of validity to problems incident to aircraft accidents involving large decelerative forces.

The "impact decelerator" (fig. 1) has proved to be a useful device in the study of impact forces, which is one aspect of linear acceleration. Early studies with this instrument on the effects of impact forces on human subjects employed the regulation restraining harness composed of seat belt and shoulder straps (1). It was found that the usual level of the subject's tolerance* was about 2000 pounds. As impacts exceeded 2000 pounds, they became increasingly painful, due in part to the relatively narrow harness area which transmits the force to the mid-abdominal and clavicular areas.

A semi-rigid, vest-type harness (Model "A") was designed (2). This harness distributed the force over the thorax and part of the abdomen and increased impact tolerance as judged by physiological measurements, subjective reactions, and the opinion of observers. Observation of subjects during impact was further aided by the use of high-speed motion pictures taken at 3000 frames per second (3). In this report additional data are presented on respiratory and circulatory changes during impact.

During the course of these experiments in which impact loads were cautiously and progressively increased, it was learned that if the duration of impact was prolonged, the configuration of the force-time curve could be changed and the peak force would be diminished (2).

Since the physiological effects of prolonged compression of the human thorax by large static loads had heretofore not been studied, and because the prolongation of impacts was contemplated, it was considered desirable also to investigate these effects.

*Tolerance is defined as that value of impact or load which produces a painful reaction.

METHODS

Fifteen impact experiments on five individuals whose ages averaged 19 years (spread 18-21), are reported, and 18 static loading tests on eight similar subjects are included. Physical examinations of these subjects conducted before and after the study revealed normal findings. The subjects were placed in a supine position on the impact decelerator, and static loads or impact forces were applied to the anterior chest and abdomen by a vest-type restraining harness and recorded by calibrated wire resistance strain gages. The duration of the force-time curve can be altered by placing springs between the weights and the rod head (4). Such impacts produced a uniform force-time curve, the peak of which ranged between 1800 and 3000 pounds. This peak was reached in approximately 50-70 milliseconds with an average total duration of 150 milliseconds.

Electrocardiogram.- Precordial leads were placed over the area of maximal apical impulse and at the pulmonic area; the indifferent electrode was applied just above and medial to the left scapula. This arrangement of "lead 4B", compares favorably with the conventional arm leads and has helped to avoid interference from skeletal muscle potential.

Ear pulse and ear opacity.- An instrument containing a photo-electric cell attached to the pinna of the ear was used to record arterial pulsations and changes in blood volume (opacity) occurring within the ear. The ear pulse provides an accurate index of the arterial pulsations in the ear and satisfactorily parallels the changes occurring in cardiac rate and rhythm demonstrated in the electrocardiogram.

Respirometer.- The respirations were detected by a thermocouple enclosed within two lightweight plastic plugs and inserted into the subject's nostrils (fig. 2). Inspiration and expiration respectively cool and warm the thermocouple wire and cause a deflection of a galvanometer. While this device does not measure actual respiratory volume it gives an index of respiratory rate and depth when the subject breathes through his nose (figs. 3,4). The rate, however, is distinguishable even when the subject breathes orally.

Radial pulse and pressure applied.- The radial pulse was recorded by means of a suitable small electrical wire strain gage. From this radial pulse record an index of pulse pressure was obtained. In some cases the pressure in pounds per square inch (psi.) was measured by a small strain gage placed between the anterior thoracic wall and the harness. After appropriate amplification these various physiological impulses were recorded with a General Electric oscillograph (Type PM 10). Another oscillograph simultaneously recorded the strain gage determinations of force applied to the various parts of the body.

Static loads.-- In the subjects tested with static loads, 550 pounds were applied to the chest and abdomen by traction on a similar harness (Model A) using a hand hoist under conditions of gradual and rapid loading. For gradual loading the total weight of 550 pounds was applied in 150-pound increments at 20 second intervals. Rapid loading was accomplished by quickly applying the total load of 550 pounds within two seconds. Precautions were taken to eliminate sudden impacts during the application of weights.

RESULTS USING IMPACTS

Electrocardiogram.-- Most of the tracings stay on the isoelectric line and the application of impact is marked by a "spike" (figs. 3,4). This "spike" is thought to be an electrical artifact introduced by the impact on the chest, and presumably is not of cardiac origin. Although electrocardiographic interpretation of "T" waves and QRS complexes cannot be evaluated when only precordial leads are employed, changes in rate and rhythm can be recorded satisfactorily. A tachycardia customarily is associated with the apprehensive anticipation of an impact (table 1). Each impact is marked by a single biphasic deflection as the force is applied. This deflection is followed by several distorted upright waves (fig. 3). These waves indicate cardiac activity probably of ventricular origin since they are reflected in the peripheral ear pulse. Approximately one second after the impact a QRS complex appears followed after two seconds by another QRS complex. The preceding P wave is almost absent. A slowed rate approximating 50 to 55 beats per minute is maintained for the next ten seconds, following which there is a gradual return to normal*. This slowing of rate also has been observed when 550 pounds of static load is applied to the chest.

In experiments exhibiting slowing to a similar degree immediately following an impact, the subject complained that the impact was painful, and experienced nausea associated with pallor. It will be noted that subject HWH (fig. 3) already had completed a forced expiration when the impact was applied. Impact occurring after complete expiration causes more discomfort than usually is experienced.

The first ten seconds following an impact usually are associated with cardiac slowing as compared with the ten seconds just prior to the dropping of the weight. Occasionally the rate may be observed to decrease 50 per cent following the impact.

*Contractions were counted for only a ten-second interval and expressed as rate per minute. The rate is changing rapidly following the impact and the value given represents that rate only during the interval counted.

Table 1.- Physiological data on impacts. Comparison of pulse rates at the beginning of each experiment, during the period of anxiety which immediately precedes the impact, immediately following the impact, and during one minute after the impact. An apprehensive tachycardia is almost invariably demonstrated before the application of the impact force.

Exp. number and subject	Impact force (lb.)	Cardiac contractions per minute*			
		Control	Immediately before impact	Immediately following impact	One minute after impact
#237 D.H.F.	1520	138	156	144	126
	2520	144	150	96	112
	2680	96	138	112	96
#240 R.J.F.	1970	96	112	78	78
	1890	108	126	90	66
#241 P.J.B.	1910	90	138	132	96
	2008	90	120	114	84
	2490	90	126	120	90
#242 H.W.H.	1800	96	108	120	84
	1632	96	120	108	96
	-	96	108	96	72
#244 M.D.D.	1812	84	84	84	72
	2950	66	112	84	54
#246 P.J.B.	2782	108	168	144	112
#255 D.H.F.	2580	84	168	150	126
#256 M.D.D.	2470	86	144	138	112
#257 P.J.B.	2460	84	114	120	78
#256 M.D.D.	2520	66	112	78	60
	2310	66	108	78	60
	2290	90	112	78	54

Table 1 (Continued)

Exp. number and subject	Impact force (lb.)	Control	Cardiac contractions per minute*		
			Immediately before impact	Immediately following impact	One minute after impact
#258 H.W.H.	2700	114	108	66	72
	2725	114	120	96	72
#260 D.H.F.	2515	---	156	150	---
	2515	156	162	114	120
	2540	138	144	126	120
	2310	126	138	132	108
#261 M.D.D.	2260	72	90	112	78
	2620	96	96	90	66
#262 P.J.B.	2818	90	120	132	96
#263 M.D.D.	1720	66	72	66	54
	1710	66	72	60	48

*Beats counted only for a ten-second period and expressed as rate per minute.

Ear pulse and opacity.-- The gradually increased ear opacity which is associated with the expiratory straining maneuver begins a few seconds before the impact. The subject develops a pronounced flush of the face and ears while carrying out this maneuver. A sharp upward deflection of the ear opacity curve, denoting an increase in ear blood volume, occurs within 50 to 120 milliseconds after the impact (fig. 4).

Respirations.-- Early in the study it was found that an impact delivered to the chest and abdomen was less distressing if it occurred during a time when the subject expired forcibly against a partially closed glottis. A standard "ready" period of two seconds was used therefore before each drop. During this interval the subject inspired deeply and started a slow forced expiration to coincide with the impact. The impact applied at that time forced the remaining air out of the lungs. A period of relative apnea lasting approximately six seconds occurred and was followed by fairly deep and rapid respirations, slower than previously recorded (fig. 4). No other consistent respiratory changes were observed.

RESULTS USING STATIC LOADS

Rapid loading.-- As the load is applied the chest and abdomen are fixed by the resulting pressure. Respiratory changes under the stress

of load is detected by the respirator. Respirations usually are observed to be diminished or absent. Marked flushing of the head and neck occurs, which is followed shortly by a moderate cyanosis. The electrocardiogram shows an average slowing of 20 beats per minute during the loading; a bradycardia of less than 50 beats per minute is present in four cases (table 2). Although the average of the heart rates before loading is 90 contractions per minute, the heart rates after the loads are removed average 100 contractions per minute*. This augmentation of rate is considered significant. Changes in the electrocardiographic complexes are not interpretable (fig. 5). The radial pulse recording shows a rapid decrease in pulse pressure. The character of the radial pulse is progressively altered; its amplitude diminishes until the dicrotic notch is barely discernible when loads of 300 pounds are exceeded. Ear opacity is increased. The configuration of the ear pulse shows a diminished amplitude similar to that shown by the radial pulse. When it is impossible to see the radial pulse, auscultatory sounds during blood pressure determinations are not detectable.

Upon removal of the load, the first three auscultatory sounds appear immediately. The fourth sound is not heard until four to six seconds have elapsed. During this period what may be the dicrotic notch appears to be abnormally placed within the pulse configuration (fig. 6). During the obliteration of the pulse under load, the blood pressure cannot be measured by the arm cuff method. For this reason and because of the artifacts introduced during the loading, the blood pressure measurements are not reliable and therefore are not reported.

The pressure at the right pectoral area as measured by the electrical wire strain gage method averaged 2.7 psi. in five experiments. Subjects have tolerated rapid loading of 550 pounds for periods varying from 20 to 40 seconds. One individual (PJB) was able to withstand a total load of 550 pounds for 80 seconds (table 2).

Gradual loading.— Essentially the same physiological phenomena that occur upon gradual loading also occur with rapid compression (table 2). Since the subject must support the weights for a longer period of time when they are added at 20-second intervals, the tolerable duration for the total weight of 550 pounds is diminished accordingly. The changes are slower than were observed with the rapid loading. The return to normal, following removal of the load, occurs as quickly as that observed under rapid loading.

*Pulse counted during a six-second interval and is expressed as rate per minute. This rate represents only those during the six-second period and not during the entire minute.

Table 2.- Physiological data on one series of volunteers during static loading (rapid) of 550 pounds

Subject	Duration of static load on chest (sec.)	Heart contractions per minute*			Respiration	Ear opacity	Radial pulse
		Immediately before load is applied	During 550-lb. static load	Immediately after load is removed			
MMD	28	60	60	100	ASD**	Increased	Absent
MDD	28	80	60	90	AMD***	"	"
MDD	10	70	50	80	Absent	"	"
MMD	18	70	40	90	AMD	"	"
HWH	23	100	80	100	ASD	"	"
DF	19	100	90	120	ASD	"	AMD***
DF	23	110	70	110	AMD	"	AMD
HWH	16	80	40	90	Absent	"	Absent
HWH	20	70	40	80	"	"	"
MDD	30	70	40	100	"	"	"
PJB	60	120	100	120	AMD	"	ASD**
PJB	80	90	90	120	AMD	"	AMD
RED	22	110	80	110	ASD	"	AMD
JGC	20	90	80	70	AMD	"	ASD
FER	15	90	80	100	AMD	"	AMD
FER	30	100	70	110	AMD	"	AMD
RRD	15	70	60	60	AMD	"	Not re-corded
FER	18	110	90	110	AMD	"	"
Mean heart rate		88	68	98			

*Pulse taken during six-second intervals immediately before and after weight was applied and immediately after weight was removed, expressed as rate per minute.

**ASD - Amplitude slightly diminished.

***AMD - Amplitude markedly diminished.

DISCUSSION

The biophysical technics which have been utilized are almost identical with those developed for use in the study of radial acceleration; used with care they have yielded accurate and consistent results. With further experience in their employment and when precise standardization is possible, they should provide a quantitative analysis of a variety of biological responses elicited by linear accelerative forces.

Since volunteers had been used in these experiments every effort was made to avoid injury to them. Thus, most of the impacts sustained have been well below the injury-producing threshold. With the vest type harness most subjects have shown relatively minor discomfort even when 3000-pound impacts were sustained. Occasionally, however, such a force caused very painful reactions, especially when the subject would not synchronize the forced expiratory maneuver with the onset of impact.

There is an appreciable period, averaging two to three seconds, after impact during which the subject is pinned down by a static load of 165 pounds or more on his chest and abdomen. This period of static loading makes inspiration difficult, and together with the shock of the impact probably accounts for the apnea observed.

The decreased heart rate which follows an impact administered to a relatively unyielding thorax in cats has been reported (5). It is probable that such a sudden slowing of the pulse is due to direct vagal stimulation, either by sudden, increased pressure on the aortic arch, carotid sinus, or even via the vagus fibers in the partially distended pulmonary alveoli. Frequently associated with this slowed rate are a few dropped or incomplete heart beats which occur immediately after the impact.

The increase in ear opacity during the two seconds before impact is probably due to increased intrathoracic pressure leading to excessive filling of the vessels of the head and neck. The recorded sudden upward deflection of the ear opacity, which follows the impact by 100 milliseconds, is believed to represent a vascular transmission wave due to sudden compression of the chest and abdomen.

The major physiological changes are probably caused by the mechanical effects of external compression of the thorax and upper abdomen. During the load period external movements of the thoracic wall have not been observed (table 2), although the respirometer recorded small rapid excursions in most cases. These recordings are probably a reflection of diaphragmatic movements in an attempt to breathe with a fixed chest.

The flushing of the head and neck is probably caused by an increased venous engorgement due to retrograde flow from the thoracic pool and the inability of the returning blood to enter the thorax. The cyanosis which later becomes superimposed upon this flushing may be attributed to stasis.

The relative bradycardia under load conditions may be explained by direct vagal stimulation resulting from an increased intrathoracic pressure. The basic mechanism causing this bradycardia awaits further studies.

The changes in the peripheral pulse configuration may be explained on a mechanical basis. The available blood in the thoracic vascular pool can be expressed peripherally under load. This increased volume of blood may cause a temporary distension of the arterial wall, thereby altering transmission. This changes the configuration of the peripheral pulse. It is probable that other mechanisms also are operative.

SUMMARY

1. Biophysical technics have been utilized to study the effects of impact forces and of static loading on volunteers. These technics include the use of the electrocardiograph, thermocouple respirometer, and a photoelectric cell for determining ear pulse and ear opacity. Electrical wire strain gages were employed for pulse and pressure determinations.
2. A static load of 550 pounds was applied to the anterior chest and abdomen of eight male volunteers by the use of traction on a vest type restraining harness. The results of 18 such loadings are reported.
3. With this static load, thoracic respirations diminish or cease, pulse pressure approaches zero, the ear volume increases, and a moderate relative bradycardia is demonstrable.
4. Impact loads varying from 1500 to 3000 pounds produce a rapid increase in ear opacity 50 to 90 milliseconds after the impact. This increase in ear volume probably represents a vascular transmission wave initiated by sudden abdominal and thoracic compression.
5. Impacts usually are preceded by a rapid and shallow respiratory pattern. Apnea occurs immediately after the impact followed within five to ten seconds by deep and slow respirations.
6. Forced expiration against a partially open glottis has been found to be the best method to protect subjects during impacts.

7. Impacts causing little or no discomfort do not materially alter the electrocardiographic pattern. Impacts causing minor distress are followed by a sudden transient slowing of the heart rate of from 10 to 20 beats per minute. With more painful impacts, an immediate bradycardia of 55 beats per minute has been observed. This bradycardia probably is due to direct vagal stimulation although other mechanisms may be involved.

8. The radial and ear pulses reflect the changes in heart rate seen in the electrocardiograph.

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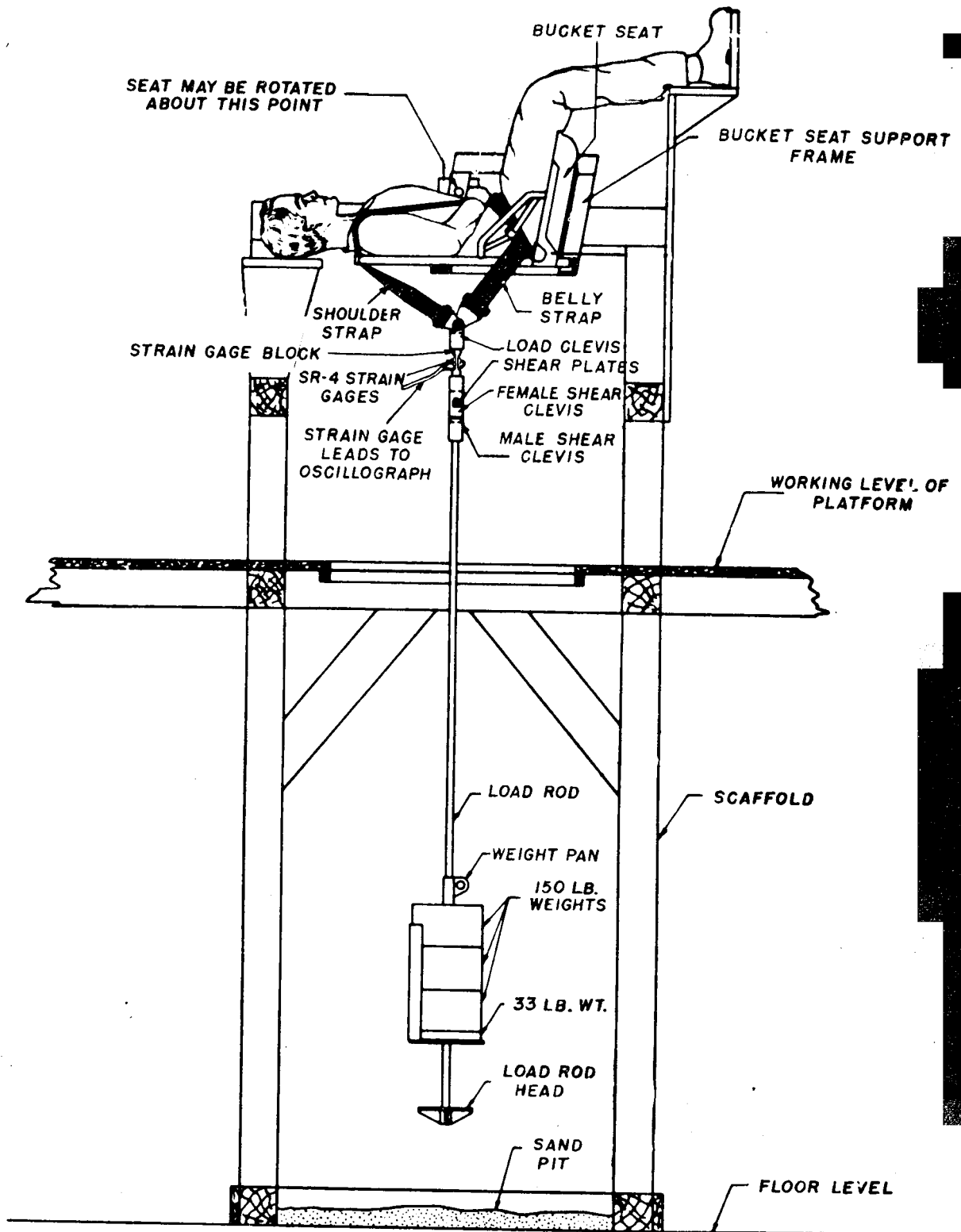


Figure 1.-- Diagram of the impact decelerator. The weights are applied and removed by a hoist for static loading



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Figure 2.—Photograph of subject wearing thermocouple respirometer in-
closed in light plastic plugs

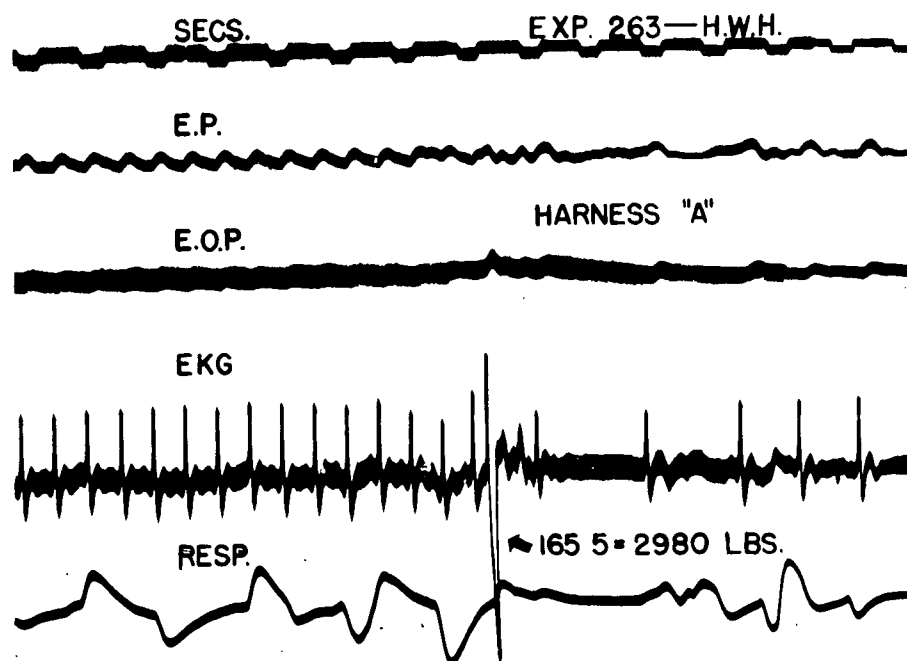


Figure 3.—Oscillographic record obtained when subject H.W.H. sustained 2980 pounds impact force. The time of impact is indicated by an arrow. Note immediate bradycardia and apnea following impact.

SECS.

EXP. 262—P.J.B.

988

E.P.

HARNESS "A"

E.OP.

E.C.G.

RESP.

165/5' = 2818 LBS.

Figure 4.—An impact of 2818 pounds with a semi-rigid vest harness (Model A) occurs at the arrow. Resp.—Inspirations are downward and expirations are upward. Note the temporary cessation of respiration soon followed by a deep irregular pattern. E.C.G.—Electrocardiogram remains on the isoelectric line despite the impact. E.OP.—the ear opacity shows a slow, gradual increase a few seconds prior to the impact. This is caused by the forced expiration of tensing in anticipation of the blow as confirmed by the respiratory pattern. Almost simultaneously with the impact, there is an abrupt increase in opacity which gradually returns to normal. This is evidence of a wave transmitted to the ear by way of the vascular system. E.P.—the ear pulse reflects the changes that occur in the electrocardiogram and the ear opacity.

TIMER

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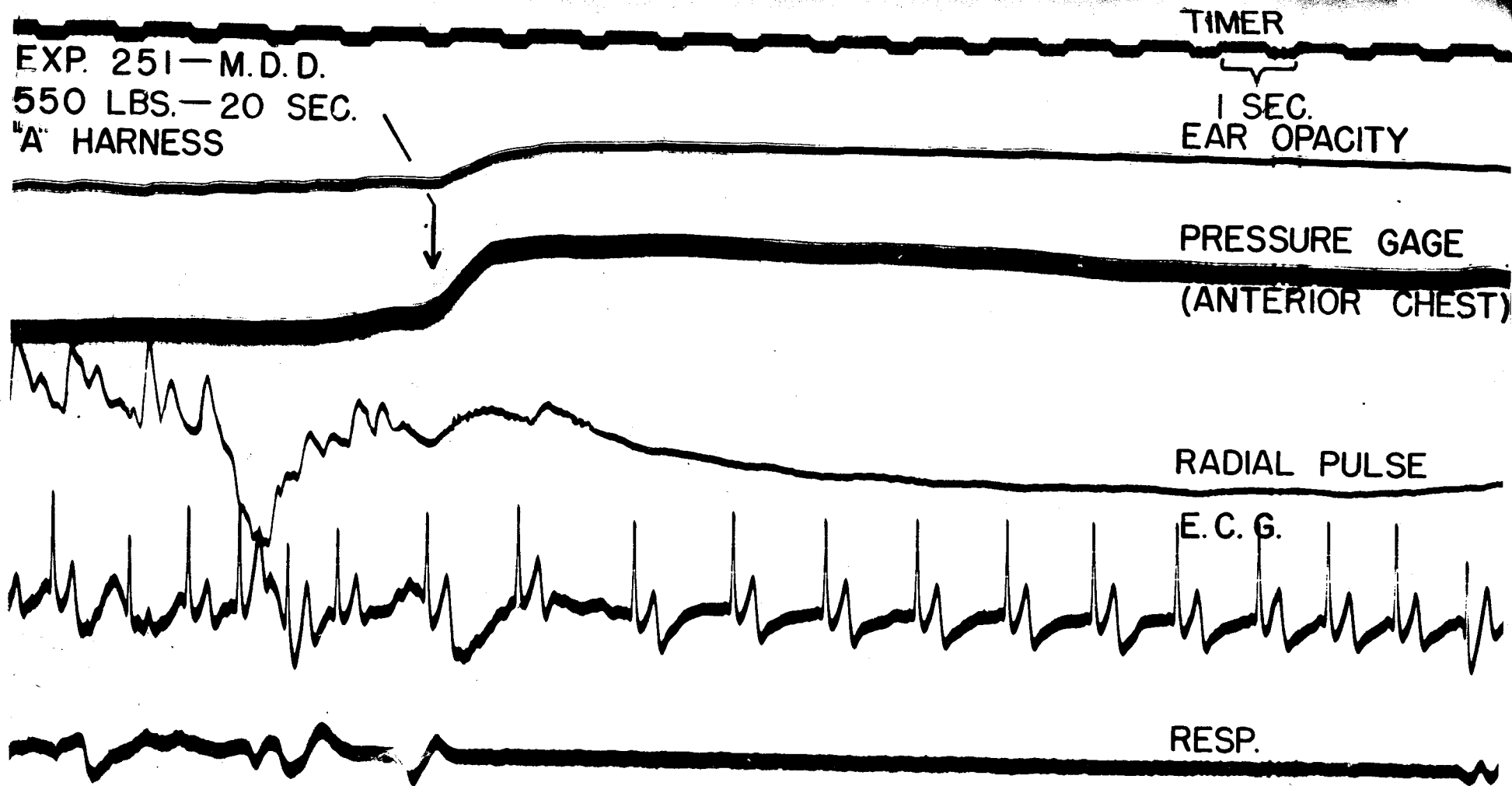


Figure 5.—Physiological effects of a rapidly applied static load of 550 pounds to the thorax and upper abdomen of a human. A pressure of 2.8 (psi) was recorded on the right pectoral area by means of a strain gage. Note cessation of respiration, bradycardia, an increase in ear opacity, and a diminished amplitude of radial pulse followed by complete loss of pulse.

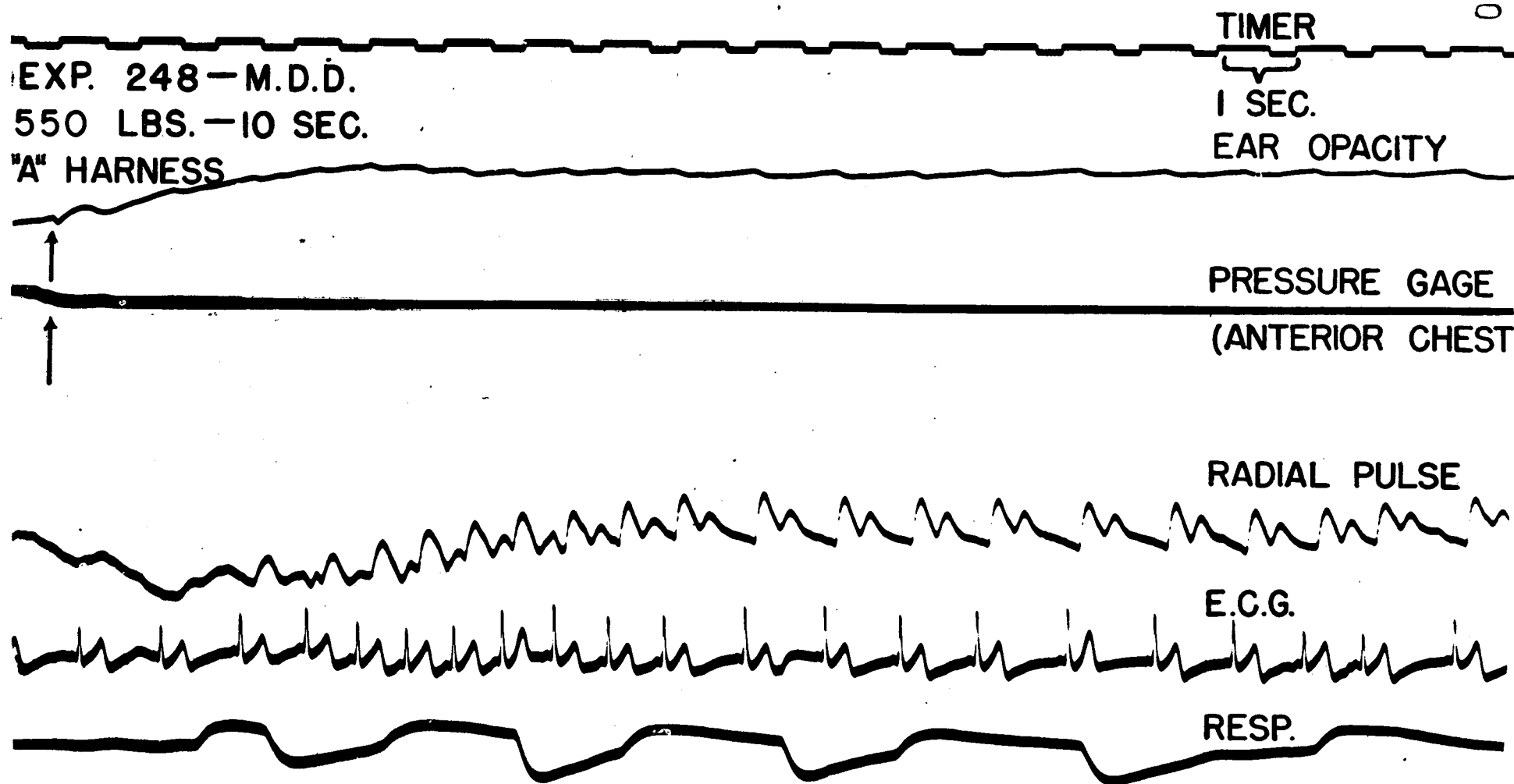


Figure 6.—Physiological effects upon removal of 550 pound load from the thorax and upper abdomen of a human. Note restoration of respiratory pattern, tachycardia, return of ear opacity and radial pulse configuration to normal, and the change in position of the dirotic notch.